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ABSTRACT

The paper refutes the long held belief that Down's syndrome is the result of maternal age and maternal etiology. The author cites new evidence which demonstrates that Trisomy-21 (the presence in the chromosome of an extra arcocentric chromosome resulting from non-disjunction), the major cause (95% of the cases) of Down's syndrome, can originate in the egg or the sperm. Maternal origin was indicated in 74% of the cases and paternal origin was indicated in the 26% of the cases reviewed in the paper. The author concludes with a study of 37 college texts dealing with exceptionality (published between 1970-1978) reviewed for etiological considerations of Down's syndrome. Among findings of the review was that of the 35 texts which mention Trisomy-21, 26 include information regarding maternal origin and maternal age dependency. (Author/PHB)

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PATERNAL CONTRIBUTION TO DOWN'S SYNDROME DISPELS MATERNAL MYTHS

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Abstract

Approximately 90-95% of all of the Down's Syndrome cases are due to Trisomy-21, the presence of an extra acrocentric chromoson resulting from nondisjunction. The incidence of Down's Syndrome appears to increase with advanced maternal age. Because oocytes remain in a sustained meiotic prophase from prenatal life until ovulation, it has been postulated that the chromosomes of ooctyes become more subject to nondisjunction with aging, and that Trisomy-21 is caused by faulty ova.

Modern chromosome banding techniques sometimes allow unequivocal identification of the parental origin of the extra chromosome. In a summary of published informative karytopes paternal origin is indicated in 26% of the cases. Moreover, a survey of recent statistical studies reveals that data from Denmark and Japan indicate the presence of a paternal age effect which increases sharply among fathers 55 and older.

This paper reviews 37 college texts dealing with exceptionality. Of these texts, 26 mention maternal origin and maternal age dependency in Trisomy-21; paternal origin and age dependency is mentioned by only one text. Thus, popular misconceptions that Down's Syndrome is caused exclusively by mothers' faulty eggs and advanced age are being supported in the literature of exceptionality. Awareness of recent cytological and statistical data which indicate both a maternal and a paternal origin for Down's Syndrome should be useful to parents, researchers, and professionals in human services.

Delays in the normal development of a child invariably produce insidious, nagging fears in a parent. When suspicions of abnormality are confirmed, usually by a physician, a series of typical parental reactions occurs.

There is shock and denial followed by frantic searching for proof that the child is normal (Barsch, 1968; Emde and Brown, 1978; Macmillan, 1977; Noland, 1970). Unfulfilled aspirations pose feelings of guilt, failure, and a threat to self-esteem (Antley & Harlage, 1976; Horrobin & Rynders, 1975; Solnit & Stark, 1961; Wolfensberger, 1967). Compelled to find a cause of their child's handicap, it is common for parents to invoke factors such as medical incompetence, a childhood fall, or trauma during pregnancy or parturition (Barsch, 1968; Ehlers, 1966; Macmillan, 1977).

Today, in cases of Down's Syndrome (mongolism) the time span for these reaction stages is greatly compacted. Physicians inform parents that their newborn is mentally retarded shortly after birth. Physical characteristics of the infant facilitate an early diagnosis of Down's Syndrome: eyes which slant upwards with small skin folds at the inside corners, flattened head and facial contours, a short, broad neck, and unusual dermatoglyphics. Parental disbelief and hope that their child will outgrow these features (Barsch, 1968; Ehlers, 1966; Emde & Brown, 1978; Horrobin & Rynders, 1975; MacMillan, 1977) are sharply thwarted by chromosomal analysis. Usually for parents of a Down's Syndrome child a diagnosis of mental retardation (with practically no exception) is swiftly determined (Yunis, 1977).

Unlike the mother of a child who is later identified as mentally retarded, the mother of a Down's Syndrome child has little time to prepare herself psychologically for having a mentally retarded child. She cannot evoke an unknown environmental agent causing brain damage as an etiological defense mechanism. The mother of a Down's Syndrome baby is immediately confronted with physical stigmata and then pounded by widespread and generally accepted theories that attribute the causation to an error in the formation of her ovum. If the mother is older, her anguish is intensified.

". . . the possibility of a chromosomal error . . . is known to be significantly higher toward the end of the reproductive years. All the egg cells that a woman produces throughout her life are present in immature form in her ovaries from the time she is born. As she gets older, chances increase that a mishap will occur during maturation of an egg prior to its fertilization; hence she is at increased risk of having a child with Down's Syndrome or other birth defects due to chromosomal error." (The National Foundation, March of Dimes, 1978, p. 11)

Are these widespread and generally accepted theories justified? What is the present stage of biological knowledge concerning the etiology of Down's Syndrome? How is this information being transmitted in current texts dealing with exceptional children? In this paper we review recent literature in cytology and exceptionality, seeking to answer these questions.

Human Cytogenetics

A karyotype is the chromosomal complement of an individual. The term is also used for photographs taken of human chromosomes during the mitotic metaphase and arranged in a standard sequence. In order to make a karyotype, a small sample of blood is drawn and the lymphocytes are stimulated to divide by addition of a mitotic inducer. After the cells have been cultured for several days, the drug colchicine is applied, inhibiting division of the white blood cells at metaphase.

The cells are fixed, stained, observed under a microscope, and photographed. The developed photomicrograph is enlarged, and the individual chromosomes are cut out and arranged in decreasing order of size. Sex chromosomes are designated X and Y. The non-sex chromosomes, or autosomes, are arranged in homologous pairs serially numbered 1-22 in decreasing order of length, and classified into seven groups on the basis of size and position of a constriction known as a centromere (Levitan and Montague, 1972). Within the groups, individual pairs of homologous chromosomes can be distinguished by the distinctive fluorescent banding patterns that appear when the chromosomes are stained with quinicrine mustard or other fluorescent dyes (Caspersson et al., 1971). A normal human karyotype has 46 chromosomes, 22 pairs of autosomes and one pair of sex chromosomes.

Oogenesis is the formation, development, and maturation of the female gamete or egg. Spermatogenesis is the formation, development and maturation of the male gamete or sperm. Genetically, the most important part of oogenesis and spermatogenesis is meiosis, the two cell divisions that reduce the

adult, diploid, chromosome number of 46 to the gametic, haploid chromosome number of 23.

Male meiosis begins at puberty and then continues throughout the course of the male reproductive lifetime taking approximately 70-78 days to complete a maturational cycle. In the human female, meiosis begins during embryonic life and is not completed until after the sperm has penetrated the oocyte. Women are born with their entire lifetime supply of oocytes (or future eggs); moreover these oocytes remain in an arrested stage of meiosis for between roughly 15-50 years (Baker, 1972; Monesis, 1972).

Occasionally during meiosis in the male or the female, one of the chromosome pairs fails to separate. This process is called nondisjunction and results in the formation of a sperm or egg carrying an extra chromosome. At fertilization by a normal haploid gamete, the zygote will now bear 47 chromosomes. This chromosomal condition is referred to as a trisomy. The phenotypic effects of trisomy are characterized by multiple defects involving many organs and systems.

Cytogenetic Aspects of Down's Syndrome

In humans the first example of trisomy discovered (Lejeune et al., 1959) is also the most frequently observed. It involves the smallest chromosome of the human chromosome complement, chromosome No. 21. Individuals who are trisomic for chromosome 21 display the aggregate of clinical symptoms and aberrations associated with Down's Syndrome, so it can be said that Trisomy-21 is the "cause" of Down's Syndrome. However, the cellular event that triggers the chromosomal

error (usually non-disjunction) leading to Trisomy-21 is not understood. Thus the ultimate etiology of Down's Syndrome remains elusive.

The results of normal meiosis and fertilization, and of non-disjunction leading to Trisomy-21, are shown diagramatically in Fig. 1. Meiosis reduces the diploid chromosome number of 46; fertilization restores the diploid number. When non-disjunction for chromosome 21 occurs, fertilization will produce a zygote with 47 chromosomes that will develop into a child with Down's Syndrome.

In Figure 2 is shown an actual human karyotype of a male child with Down's Syndrome, illustrating the presence of three copies of chromosome 21. In a normal karyotype, chromosome 21 would be present in only two copies, as a simple homologous pair.

The clinical anamolies of Down's Syndrome are caused by the presence of extra genetic material from chromosome 21. However, the extra genetic material is not always introduced as a simple trisomy. In some cases the extra dose of chromosome 21 may arise via a translocation (a breakage and rejoining of non-homologous chromosomes) or a mosaicism. For geneticists, mosaicism refers to an organism with more than one type of cell line, and as such, has implications for Down's Syndrome in two ways. Mosaicism may describe a Down's Syndrome individual in which only some of the tissues are trisomic or translocated; or it may refer to an unaffected parent whose germ tissues bear an occult chromosomal aberration which, when transmitted to offspring, produces Down's Syndrome. Down's Syndrome becomes hereditary when a certain type of translocation occurs in a

parent (a "balanced" translocation) or in the second form of mosaicism.

By far the largest percentage of Down's Syndrome cases (approximately 95%) are simple Trisomy-21 arising de novo via meiotic nondisjunction. Estimates of the incidence of the other forms of chromosomal aberration leading to Down's Syndrome vary somewhat from authority to authority, but all are low. A thorough review by Cohen et al (1977) attributes only 3-4% of all cases to translocations and other structural aberrations. Penrose (1966) estimates about 3% of mongols carry translocations but "as many as 10 percent might come from mosaic mothers." Among the Down's Syndrome offspring of younger mothers the frequency of translocations may be higher than in the total population. Deye (1977) reviewed this literature and reports an incidence of 2-9% for translocation mongols from 11 studies on younger mothers.

The issue of maternal age is a complex one, and will be discussed in more detail below (see section on "Maternal Age Effect"). At this point suffice to say, that the combination of numerous statistical studies correlating increased maternal age with increased incidence of Down's Syndrome, and the cytological knowledge that meiosis in the human female can be prolonged for decades, led to the seemingly reasonable assumption that the de novo non-disjunction events leading to Down's Syndrome took place, with only rare exceptions, in the formation of the egg, not the sperm. Only recently has it been possible to test this seemingly reasonable assumption.

The distinctive fluorescent Geimsa and other banding patterns that have been used to distinguish autosomes in different chromosomal groups within the human karyotype may also, in some cases reveal individual differences among people. These distinctive banding patterns are called "chromosome polymorphisms" and are inherited in a Mendelian fashion. In cases where there is a recognizable polymorphism for chromosome 21 in either the mother or father of a Down's Syndrome child, the parental origin of the extra chromosome can be determined by making karyotypes of the Down's Syndrome child and both parents. Currently the technique is limited by the frequency of detectable polymorphisms. When quinicrine fluorescence has been used to study polymorphisms for chromosome 21, distinctive features have been found in between 1 and 25% of the karyotypes; Verma et al (1977) have reported 50% detectable polymorphisms using acridine orange. In Table 1 we have summarized the major published studies which have used chromosome polymorphisms to determine the parental origin of the non-disjunction leading to Trisomy-21.

Of the first four reports utilizing fluorescent banding techniques in order to determine parental origin of Trisomy-21, three showed maternal origin (Licznerski and Lindsten, 1972; Mutton, 1973; and Robinson, 1973) while a fourth was uninformative (Smith and Sachdewa, 1973). The first reports of a maternal or paternal input quickly followed (Punnett and Kistenmacker, 1973; Sasaki and Hara, 1973; Uchida, 1973). Subsequent larger studies have confirmed that the extra chromosome in Trisomy-21 may originate in either the mother or the father. Of the cases reviewed a total of 115 or 33%

were informative as to parental origin of the extra chromosome; of the informative cases 85 or 74% indicate maternal origin and 30 or 26% indicate paternal origin. It can be expected that future surveys encompassing larger samples and improved staining techniques will change these figures, but for the present there is an important inference. The extra chromosome in Trisomy-21 is not exclusively of female origin, and the "reasonable assumption" concerning maternal age effects warrants re-examination.

Maternal Age Effect

The increased incidence of Down's Syndrome among the offspring of older parents, and among last born children has been recognized historically (Brousseau, 1928). Since maternal age, paternal age, and birth order are highly correlated, early observers did not attempt to distinguish between their relative importance until two classic papers published in 1933 presented statistical evidence indicating that maternal age was the principal variable, with neither paternal age nor birth order being significant factors (Jenkins, 1933; Penrose, 1933). Numerous studies have affirmed a correlation between advancing maternal age and the incidence of Down's Syndrome births (Smith and Berg, 1976) and it has become standard practice in a variety of disciplines--medicine, education, psychology--to present histograms and tables of relative risk of Down's Syndrome as a function of maternal age, without including data on paternal age or birth order. Lilienfeld and Benesch (1969) summarized 13 major studies from different countries which provided incidence rates for different maternal age groups.

They concluded that, in general, the "data indicate that the risk of mongolism remains practically constant up to 30 years, after which it continues to increase during the remainder of the reproductive period. . . ." (Lilienfeld & Benesch, 1969).

Several investigators have pointed out that the distribution of Down's Syndrome births, when plotted according to maternal age, presents a bimodal distribution. Penrose (1967) has postulated that this bimodal distribution reflects two categories of Down's Syndrome: Class A or maternal age-independent (< 35) and Class B or maternal age-dependent (> 35). Sigler et al (1965) reject this classification, and suggest that the early so-called "age-independent" peak may be an artifact, merely mirroring the peak in the distribution of births in the child-bearing population in general.

Whether or not one accepts the formal categorization of a maternal age-independent and a maternal age-dependent group, there is no question that the percentage of births to women under 35 is increasing in the U.S.A. and most developed countries. As the average age of child-bearing women drops, the number of Down's Syndrome births among younger women increases. When Penrose made his original analysis of the bimodal distribution, he estimated that 22 percent of the cases were maternal age-independent; in a later analysis of maternal age distributions collected from 11 countries, he estimated that 33-50 percent were in the age-independent class (Lilienfeld, 1969). A very recent summary of studies from Canada, Japan, Denmark and the U.S.A. concluded that "by the

early 1970s, only about 20 percent of the infants with the Down Syndrome were born to women 35 years of age and older" (Holmes, 1978).

The change in the maternal age distribution of most Down's Syndrome births points out a need for revisions of tables of relative risk currently used in genetic counseling. Both maternal and paternal ages must be considered. This plea is substantiated by cytological studies which ascribe paternal origin for the extra chromosome in Down's Syndrome in about 25 percent of the cases (see Table 1). Where parental age was recorded in these chromosomal surveys, "comparison of maternal age in the two groups showed younger maternal ages in the group of paternal non-disjunction than in the group of maternal non-disjunction" (Stene et al., 1977). Moreover, these cytological findings have evoked a re-examination of the early statistical studies concerning maternal and paternal age.

Recently Danish workers applied newly developed statistical methods to study 218 Trisomy-21 subjects and 5,633 controls to explore the relative incidence of Down's Syndrome with advancing paternal age, with maternal age controlled. A marked increase in Down's Syndrome births was found among fathers 55 years and older (Stene et al., 1977; Stene and Stene, 1977).

A Japanese study (Matsunga et al., 1978) compared paternal age distribution of 1279 Trisomy-21 subjects born during 1952-68 with the corresponding distribution for the general population. The results of the Japanese study are similar to those in the Danish study; the relative incidence of Down's Syndrome with

advancing paternal age appears to increase slowly for fathers up to age 45-49. It increases sharply among fathers 55 and over.

The need to consider the age contributions of both parents has recently been demonstrated statistically by Abroms et al. (1979). Data was originally collected by Sigler et al., (1965) in a Baltimore study of Down's Syndrome children in which a control group of non-Down's subjects was selected by matching the birth certificates and maternal age. This study was later extended (Cohen et al., 1977), resulting in a total of 365 Down's Syndrome cases and an equal number of matched controls; their analyses of these data did not detect a statistically significant relationship between paternal age and Down's Syndrome (Sigler et al., 1965; Cohen et al., 1977). However, recent analysis of these pooled data, using multiple linear regression, suggest that the independent effects of paternal age, across maternal age categories, contribute to the prediction of Down's Syndrome births in a manner similar to maternal age (Abroms et al., 1979). This re-interpretation of the Baltimore data had been predicted by Mantel and Stark (1967) in a letter where they emphasized: "in view of the high intercorrelation between maternal and paternal age, paternal age cannot, for the data considered, account significantly for the variation in Down's Syndrome incidence once the apparent effect of maternal age has been taken into account."

In summary, advances in both cytology and statistics are necessitating a re-evaluation of the widely accepted assumption that Trisomy-21 is usually caused by non-disjunction in aging oocytes.

Survey of Texts Dealing with Exceptional Children

In the present study thirty-seven college texts dealing with exceptionality published between 1970-78 were reviewed for etiological considerations of Down's Syndrome. Among these, thirty-five include mention of the most prevalent chromosomal etiology, an "extra" chromosome or Trisomy-21. Within this group twenty-six texts include information regarding maternal origin and maternal age dependency: One text (Cleland, 1978) cautiously considers the notion of paternal age dependence:

> Although the age of mothers has been given a far more important part in the problem of mental retardation, there is some evidence that overage fathers may also help to create "high risk of retardation" offspring (p. 228).

A comparison of Tables 2, 3 and 4 indicates that, overall, texts addressed exclusively to the exceptionality of mental retardation present a more complete coverage of the chromosomal etiology found in Down's Syndrome. As shown in Table 2, thirteen of the fourteen texts on mental retardation give descriptive information regarding one or more of the specific chromosomal categories previously mentioned. None of the texts in Table 2 refer to the vague category of "Extra" chromosome.

The category of translocation is basically discussed in one of two ways: either parental origin is not mentioned or the possibility of a translocation occurring in the germ cell of either parent is stated. Because mosaicism occurs in developing embryo, it should be noted that eight texts leave parental origin unspecified in the discussion of this chromosomal

aberration. It is within the category of non-disjunction that preponderant references to maternal origin and maternal age dependency exist.

Survey texts in Special Education, as indicated by Table 3, show pervasive reporting of maternal origin, and maternal age dependency across chromosomal categories. Six of the texts employ the vague category of "extra" chromosome. Examination of the combined categories of non-disjunction and "extra" chromosome indicates that eleven of the fourteen Special Education texts in Table 3 attribute maternal origin and maternal age with the most prevalent pathology in Down's Syndrome. A review of the literature also suggests exclusive female origin and age dependency in three of the seven texts describing the pathology of translocation. Six of the fourteen texts make reference to mosaicism, two of which infer maternal origin, age dependent.

Each of the abnormal psychology texts reviewed in Table 4 indicate maternal origin and maternal age dependence for either non-disjunction, "extra" chromosome, or Down's Syndrome in general. In only one instance, among the four chromosomal categories, is paternal origin noted.

Thus, it appears that three current bodies of literature dealing with the exceptionality of Down's Syndrome have supported popular misconceptions that Down's Syndrome is caused exclusively by a mother's faulty egg and that Down's Syndrome is related solely to advancing maternal age.

Discussion

Unlike most mothers of mentally retarded children, the mother of a Down's child is typically informed of her child's condition during a highly labile period, usually within 72 hours after delivery. The mother of a Down's child has little time to prepare herself psychologically for having a mentally retarded child.

Widespread popular notions and myths of exclusive maternal etiology and maternal age dependency surround the mother of a Down's Syndrome baby. The behavioral sciences literature of exceptionality is replete with similar and incomplete explanations of the cause of Down's Syndrome. Such reporting is frequently embellished with additional credibility by the inclusion of impressive histograms and charts showing increasing Down's Syndrome birth rates relative to advancing maternal age.

Why have not the effects of paternal age been given similar consideration? In the classic monograph of Penrose (1933) which attempted statistically to explore the paternal age effect, only partial correlations were obtained for the relationship between the maternal age and Down's Syndrome births. In later case-control studies (Sigler et al., 1965; Cohen et al., 1977) conducted in Baltimore, Down's Syndrome and control subjects were matched on maternal age. The high correlation (.847) between mothers' and fathers' ages in both index and control cases appears to have masked the independent effects of paternal age. Attempts were made in 1977 by the present authors to obtain parental age data of Down's Syndrome

births from HEW (National Center for Health Statistics; Office for Maternal and Child Health; Genetics Program of the National Institute of General Medical Sciences; Mental Retardation and Developmental Disabilities Branch, National Institute of Child Health and Human Development) in order to study maternal and paternal age effects nationwide. Only maternal age data had been collected, a curious if not biased finding.

Heretofore, it appears that the method of data collection, data analysis, and reporting of parental age effects in Down's Syndrome births have precluded a more comprehensive understanding of the paternal age effect.

Consideration of both paternal age and paternal origin has also been restricted by medical knowledge of the "aging ova"--in the female eggs take 15-50 years to complete the meiotic cycle, while in the male spermatogenesis is accomplished within two months. Even the discovery of Trisomy-21 did not diminish the notion of the "aging ova" since it was assumed that non-disjunction occurred only during oogenesis and not during spermatogenesis. It is only recently through the study of chromosomal polymorphism that both maternal and paternal origin of Trisomy-21 can be demonstrated.

To date, the statistical and cytological research reviewed in this article has been limited primarily to the medical sciences literature. Hopefully an explication of these findings within the literature of exceptionality will allow parents, researchers, and professionals in human services to broaden their perspective on the etiology of Down's Syndrome.

A CHROMOSOMAL ERROR (NONDISJUNCTION) IN THE FORMATION OF SPERM OR EGG CAN LEAD TO TRISOMY-21. Heavy lines indicate normal fertilization and development.

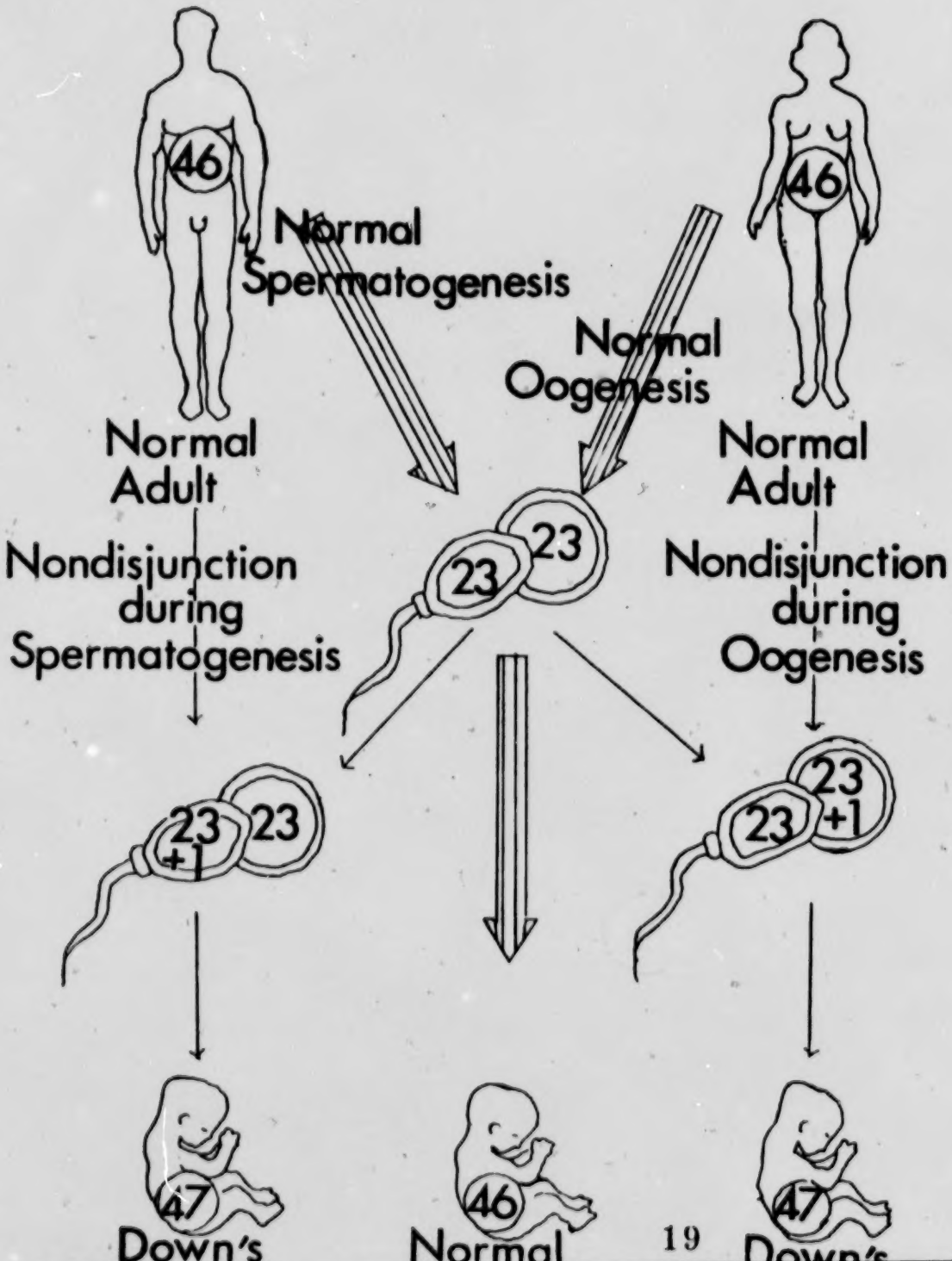

Figure 2

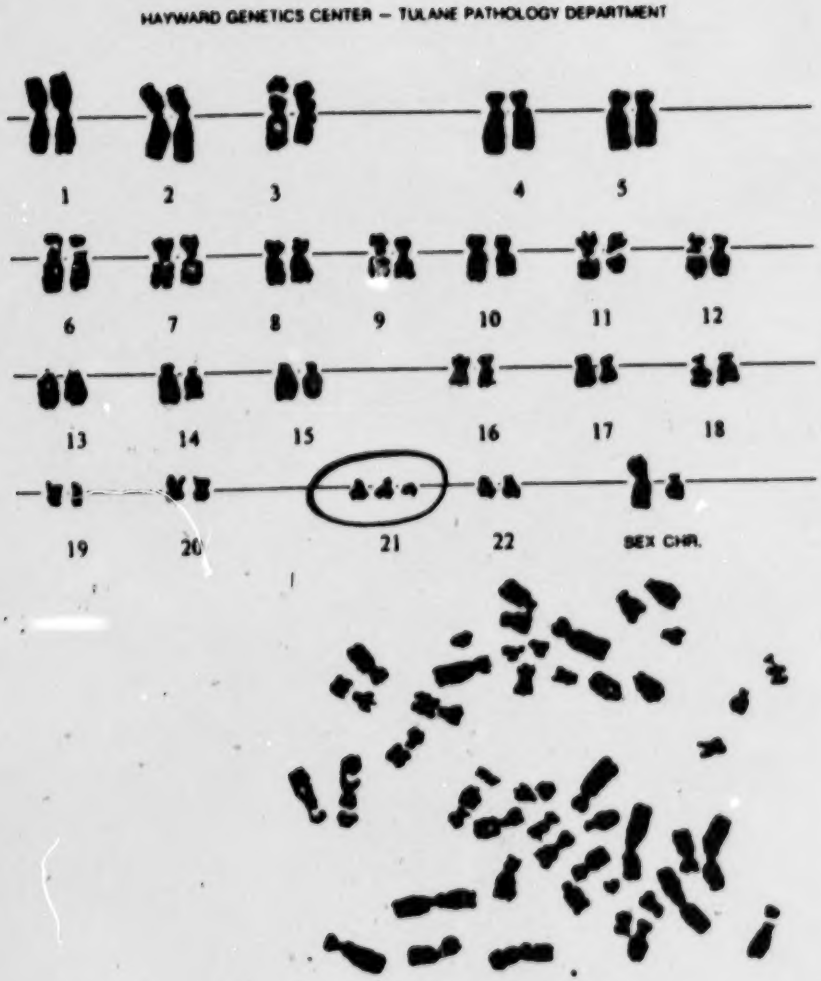
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TABLE 1

Published Reports of Origin of Extra Chromosome in Trisomy-21 Using Chromosome Polymorphisms

Citation	Informative		Uninformative	Total
	Maternal	Paternal		
Bott, Sekhon & Lubs, 1975	3	4	52	59
Giraud, Mattei, & Mattei, 1975	3	0	29	32
Hara & Sasaki, 1975	5	1	24	30
Licanerski & Lindsten, 1972	1	-	-	1
Magenis et al., 1977	24	7	11	42
Mikkelsen, Hallberg, & Poulsen, 1976	11	5	24	40
Mutton, 1973	1	-	-	1
Punnett & Kistenmacher, 1973	1	1	18	20
Robinson, 1973	4	0	8	12
Sasaki & Hara, 1973	-	1	-	1
Scmidt, Dar, & Nitowsky, 1976	22	1	23	46
Smith & Sachdeva, 1973	0	0	20	20
Uchida, 1973	-	1	-	1
Varma & Dosik, 1978	-	1	-	1
Wagenbickler et al., 1976	10	8	20	38
Total	85	30	229	344

TABLE 2

Description of Down's Syndrome Chromosomal Etiology and Parental Age Relationship Covered by 14 Texts on Mental Retardation

Texts	Descriptions of Chromosomal Etiology				
	Non-Disjunction Trisomy 21	Translocation	Mosaicism	"Extra" Chromosome	No Chromosomal Etiology Mentioned
Baroff, G. (1974)	MAD, P	M, P	NS	-	-
Carter, C. (1975)	MAD	M, P	NS	-	-
Clark, A. and Clarke, A.D.B. (1974)	MAD	MAI, PAI, PAD	NS	-	-
Cleland, C. (1978)	MAD, PAD	M, P[1]	M, P[1]	-	-
Haring, N. and Brown, L. (1977)	-	-	-	-	MAD
Horrobin, J.M. and Rynders, J. (1975)	MAD	NS	NS	-	-
Jordan, T.E. (1976)	MAD	NS	M	-	-
Kaufman, J. and Payne, J. (1975)	MAD, P	NS	NS	-	-
Kolstoe, O. (1972)	NS	NS	NS	-	-
MacMillan, D. (1977)	MAD, P	MAI, P	MAD	-	-
Mittler, P. (1977)	MAD	-	-	-	-
Neisworth, J. and Smith, R. (1978)	MAD, PAI	MAD, PAI	-	-	-
Robinson, N. and Robinson, H. (1976)	MAD, PAI	M, P	NS	-	-
Smith, D. and Wilson, A. (1973)	MAD, P	MAI, PAI	NS	-	-
Total	13	12	11	0	1

M = Maternal Origin
P = Parental Origin
MAD = Maternal Origin, Age Dependent
PAD = Paternal Origin, Age Dependent
MAI = Maternal Origin, Age Independent
PAI = Paternal Origin, Age Independent
NS = Non Specified Parental Origin

1 - See page 12.

TABLE 3

Description of Down's Syndrome Chromosomal Etiology and Parental Age Relationship Covered by 14 Texts on Special Education

Texts	Description of Chromosomal Etiology				
	Non-Disjunction Trisomal 21	Translocation	Mosaicism	"Extra" Chromosome	No Chromosomal Etiology Mentioned
Bradley, R.C. (1978)	-	-	-	NS	-
Dunn, L. (1973)	MAD	MAI, P	NS	-	-
Furneaux, B.(1970)	-	-	-	-	MAD
Gearhart, B.R. (1972)	-	-	-	NS	-
Guilliford, R. (1971)	-	-	-	MAD	-
Hallahan, D. and Kauffman, J. (1978)	MAD	MAD	MAD	-	-
Haring, N. (1974)	-	MAD	-	MAD	-
Hewett, F. and Forness, S. (1977)	MAD	MAD	MAD	MAD	-
Kirk, S. (1972)	-	NS	NS	NS	-
L'Abate, L. and Curtis, L.T. (1974)	MAD, P	MAI, PAI	NS	-	-
Meyer, E. (1978)	MAD	-	-	-	-
Ross, A. (1974)	MAD	-	-	-	-
Smith, R.M. and Neisworth, J.T.(1975)	MAD, P	-	-	-	-
Telford, C.W. and Sawrey, J.M. (1977)	MAD	MAI, PAI	NS	-	-
Total	8	7	6	6	1

M = Maternal Origin
MAD = Maternal Origin, Age Dependent
MAI = Maternal Origin, Age Independent
P = Paternal Origin
PAD = Paternal Origin, Age Dependent
PAI = Paternal Origin, Age Independent
NS = Non Specified Parental Origin

TABLE 4

Description of Down's Syndrome Chromosomal Etiology and Parental Age Relationship Covered by 9 Texts on Abnormal Psychology

Texts	Description of Chromosomal Etiology				
	Non-Disjunction Trisomal 21	Translocation	Mosaicism	"Extra" Chromosome	No Chromosomal Etiology Mentioned
Coleman, J. (1975)	-	-	-	MAD	-
Davison, G. and Neale, J. (1974)	MAD	MAI	-	-	-
Harmes, E. (1971)	-	-	-	-	MAD
Nathan, P. and Harris, S. (1975)	MAD	-	-	-	-
Page, J. (1975)	MAD	-	-	-	-
Sahakian, W.S. (1970)	MAD, P	-	-	-	-
Sarason, I. (1976)	-	-	MAD	MAD	-
Suinn, R. (1970)	MAD	-	NS	-	-
Ullman, L. and Krasner, L. (1975)	MAD	-	-	-	-
Total	6	1	2	2	1

M = Maternal Origin
MAD = Maternal Origin, Age Dependent
MAI = Maternal Origin, Age Independent
P = Paternal Origin
PAD = Paternal Origin, Age Dependent
PAI = Paternal Origin, Age Independent
NS = Non Specified Parental Origin

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